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Medical Information

Intestinal Gas

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LEVITT AND COLLEAGUES, in a series of illuminating articles published over the past five years, have elucidated the source and composition of intestinal gas, and examined its role in the production of abdominal pain.[1-6] Since "too much gas" is an exceedingly common and frustrating complaint, this brief review has been written to further publicize the results of their studies.

Volume of Intestinal Gas

At any given time, one's small intestine contains 30 to 200 cc of gas.[1,2] The amount of flatus passed per hour is usually less than 100 cc, and over 24 hours averages 400 to 1,200 cc.[1,2] Seven control patients noted the passage of flatus 13.6 ± 5.6 times per day.[3]

Many patients with complaints of excessive gas probably have normal volumes of flatus.[4] Occasionally, however, malabsorption of certain carbohydrates, as in lactase deficiency, may lead to an increased volume of intestinal gas. One such patient excreted four to five times the normal amount of flatus and passed gas 34 times per day.[3] The symptoms were greatly improved by ingestion of a lactose free diet.

Source and Composition

Using an argon washout technique, Levitt and co-workers have determined the nature of intestinal gas.[1,2] Argon is infused into the upper small intestine, and subsequently gas is collected from the rectum and analyzed. The five major components of flatus are nitrogen (23 to 80 percent), oxygen (0.1 to 2.3 percent); hydrogen (.06 to 47 percent), methane (0 to 26 percent) and carbon dioxide (5.1 to 29 percent). Since the latter three are not found in air, they must be produced within the gut.

Hydrogen (H_2) is normally produced by bacteria in the colon acting on dietary, nonabsorbable substrates, such as carbohydrates and beans.[1,2] Certain bacteria may catabolize as well as manufacture H_2, and the final concentration in flatus represents the outcome of these two processes. In one experiment, the giving of neomycin to rats increased H_2 production, presumably by inhibiting those bacteria responsible for catabolism of H_2.[5]

Methane (CH_4) is likewise produced by bacteria within the colon. Approximately a third of adults harbor such a flora, and two thirds do not.[1] Reasons for this difference are not clear. The amount of methane in the stool is largely responsible for whether or not the stool "floats," rather than the amount of fat contained.[6] Therefore, floating stools are not necessarily indicative of steatorrhea.

Carbon dioxide (CO_2) comes from two sources. Large amounts are released when gastric acid is neutralized by bicarbonate, but most of this is absorbed in the small bowel. A second source is fermentation reactions, as with H_2, in the colon.[1,2]

Nitrogen (N_2) probably comes from both swallowed air and blood. Production of H_2, CH_4, and CO_2 in the colon lowers the partial pressure of N_2 in the colonic lumen to below that of blood leading to diffusion from blood into the lumen.[1,2]

Finally, as noted above, the oxygen (O_2) content of flatus is quite low due to O_2 utilization by colonic bacteria.[1,2]

Gas and Abdominal Pain

Using the argon infusion technique, Levitt[4] and associates found 18 patients complaining of excessive gas and abdominal pain to have similar volumes and composition of intestinal gas as measured in ten control subjects. In contrast, however, such patients complained of increased abdominal pain when the argon was being infused, and showed a significantly delayed transit of the gas from small intestine to rectum. Such results suggest "too much gas" may be secondary to disordered motility, and not increased amounts of gas per se.

Conclusions

- Of the five gases found in flatus (N_2, O_2, H_2, CH_4 and CO_2), three are produced mainly in the colon (H_2, CH_4 and CO_2), and cannot be accounted for by swallowed air.
- Colonic bacteria are very important in determining the composition of intestinal gas in that they utilize O_2 and perhaps H_2, produce

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H_2, CH_4 and CO_2, and by these actions allow for the diffusion of N_2 from blood into the colonic lumen.

- Although attention to excessive air swallowing and change in diet may help some patients with "too much gas," alteration of abnormal gut motility may be of greater therapeutic benefit.

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Pathogenesis of Hemolysis in Immune Hemolytic Anemia

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AUTOIMMUNE HEMOLYTIC ANEMIA is not an uncommon problem in clinical medicine, and when it occurs it can be quite perplexing to the practitioner. In recent years our understanding of the mechanisms involved in immune red cell destruction has advanced considerably. This discussion will attempt to summarize some of these mechanisms and relate them to clinical decisions concerning course and treatment.

Hemolysis due to immunologic mechanisms is caused by a reaction between antigenic sites on the surface of the red blood cell and circulating antibody, with or without the participation of complement. The basis for detecting the presence of such a reaction is the antiglobulin test, otherwise known as the Coombs' test.[1] Antiserum is produced by injecting rabbits with human immunoprotein, either antibody or complement. The animal makes antibody to whatever is injected. That antibody can be purified and then reacted *in vitro* with a patient's washed red blood cells (RBC). If the cells agglutinate, the test is positive. By serial dilutions the degree of positivity can be quantitated.

In a recent review,[2] Wendell Rosse has classified the Coombs' test results according to whether antibody alone is detected on the red cell, antibody plus complement, complement alone, or neither antibody nor complement. This classification is useful in understanding the pathophysiology of immune red cell destruction.

Antibody Alone on RBC

In 1967, LoBuglio and co-workers showed that red blood cells coated with IgG would bind to monocytes.[3] That this binding was inhibited by excess IgG, and more specifically by excess Fc fragments, suggested that there was a binding site on monocytes for the Fc fragment of IgG. Electronmicrography showed that the monocytes caused membrane injury to the red cells coated with IgG, causing them to become spherocytes. Spherocytes were subsequently sequestered in the spleen. In the absence of monocytes, IgG caused no membrane injury. Cells coated with IgM did not bind to monocytes.[4] Subsequent workers have shown that there are subclasses of IgG: IgG_1, IgG_2, IgG_3 and IgG_4. Monocyte receptors have been found only for subclasses IgG_1, and IgG_3.[5] IgG_2 and IgG_4 will not inhibit monocyte binding of cells coated with IgG_1 and IgG_3 but these two subtypes will inhibit each other, suggesting that there is only one binding site.[6] Radiolabeling studies have showed *in vivo* that cells coated by IgG are sequestered in the spleen.[7] The characteristic of autoimmune hemolytic anemia with a Coombs' test positive for antibody alone (the "gamma Coombs' "), therefore, is an IgG antibody, spherocytes and splenic sequestration.

Antibody Plus Complement on RBC

In 1968 Huber and co-workers[8] showed that there was a second monocyte receptor; this one for the third component of complement. That this receptor was separate from the above described

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